BRIDGEBIO

Corporate Presentation
June 2026

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The presentation contains forward-looking statements. Statements made or presented may include statements that are not historical facts and are considered forward-looking within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Words such as “believe,” “anticipate,” “plan,” “expect,” “intend,” “will,” “may,” “goal,” “potential,” “should,” “could,” “aim,” “estimate,” “predict,” “continue” and similar expressions or the negative of these terms or other comparable terminology are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words. We intend these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act. These forward-looking statements, including express and implied statements relating to the advantages of our business model and overall strategy; our speed of creating new and meaningful drugs and related impact on patients, the sustainability and velocity of our engine to deliver medicines, and our value creation potential for patients; our financial position, including our expectations regarding reaching regulatory and commercial milestones; the commercial success of Attruby/Beyonttra (acoramidis) and the safety, efficacy, and market potential of Attruby/Beyonttra; the clinical, therapeutic and market potential of our clinical development programs and our pipeline, including BBP-418, encaleret, infigratinib, and BBP-812; our expected timing for submitting New Drug Applications with the U.S. Food and Drug Administration (“FDA”) and similar submissions with foreign regulatory authorities, receiving U.S. approval and commencing commercial launch for BBP-418, encaleret and infigratinib; our anticipated interactions with and feedback from the FDA foreign regulatory authorities; our expected commercial success of BBP-418, encaleret and infigratinib; the potential for and our expected timing of submitting a Biologics License Application for BBP-812; the potential of our depleter program to reverse ATTR; the potency and safety of our product candidates; and the potential benefits of our products and product candidates, reflect our current views about our plans, intentions, expectations and strategies, which are based on the information currently available to us and on assumptions we have made. Such statements reflect the current views of the Company with respect to future events and are subject to known and unknown risks, including business, regulatory, economic and competitive risks, uncertainties, contingencies and assumptions about the Company, including, without limitation, risks inherent in developing and commercializing therapeutic products, and those risks and uncertainties described under the heading “Risk Factors” in the Company’s most recent Annual Report on Form 10-K filed with the U.S. Securities and Exchange Commission (“SEC”), and in subsequent filings made by the Company with the SEC, which are available on the SEC’s website at www.sec.gov. In light of these risks and uncertainties, many of which are beyond the Company’s control, the events or circumstances referred to in the forward-looking statements, express or implied, may not occur. The actual results may vary from the anticipated results and the variations may be material. You are cautioned not to place undue reliance on these forward-looking statements, which speak to the Company’s current beliefs and expectations only as of the date of the presentation. Except as required by law, the Company disclaims any intention or responsibility for updating or revising any forward-looking statements made or presented at the presentation in the event of new information, future developments or otherwise.

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BridgeBio is a new type of biopharmaceutical company

Clearer Biology

FROM

Expensive platforms with long lead times before proof-of-concept data

TO

Assets selected to target genetic diseases at the source — higher probability of success from day one

Faster Learning

FROM

Slow and bureaucratic decision making at the portfolio level

TO

Rapid, decentralized R&D decisions — each affiliate operates with scientific focus and autonomy

Smarter Capital

FROM

High fixed costs, limited capital sources, and incentives misaligned at the portfolio level

TO

Variable costs via shared infrastructure, diversified financing, and asset-level incentives that preserve biological focus

We have built a sustainable, high velocity engine to deliver hope and medicines to the communities that we serve

> 9,500
individuals impacted by our therapies

Obtained Approval for 3 Medicines

- Attruby™ (acoramidis) 356 mg tablet
- Nulibry® (fosdenopterin) for injection
- TRUSELTIQ™ (infigratinib) capsules

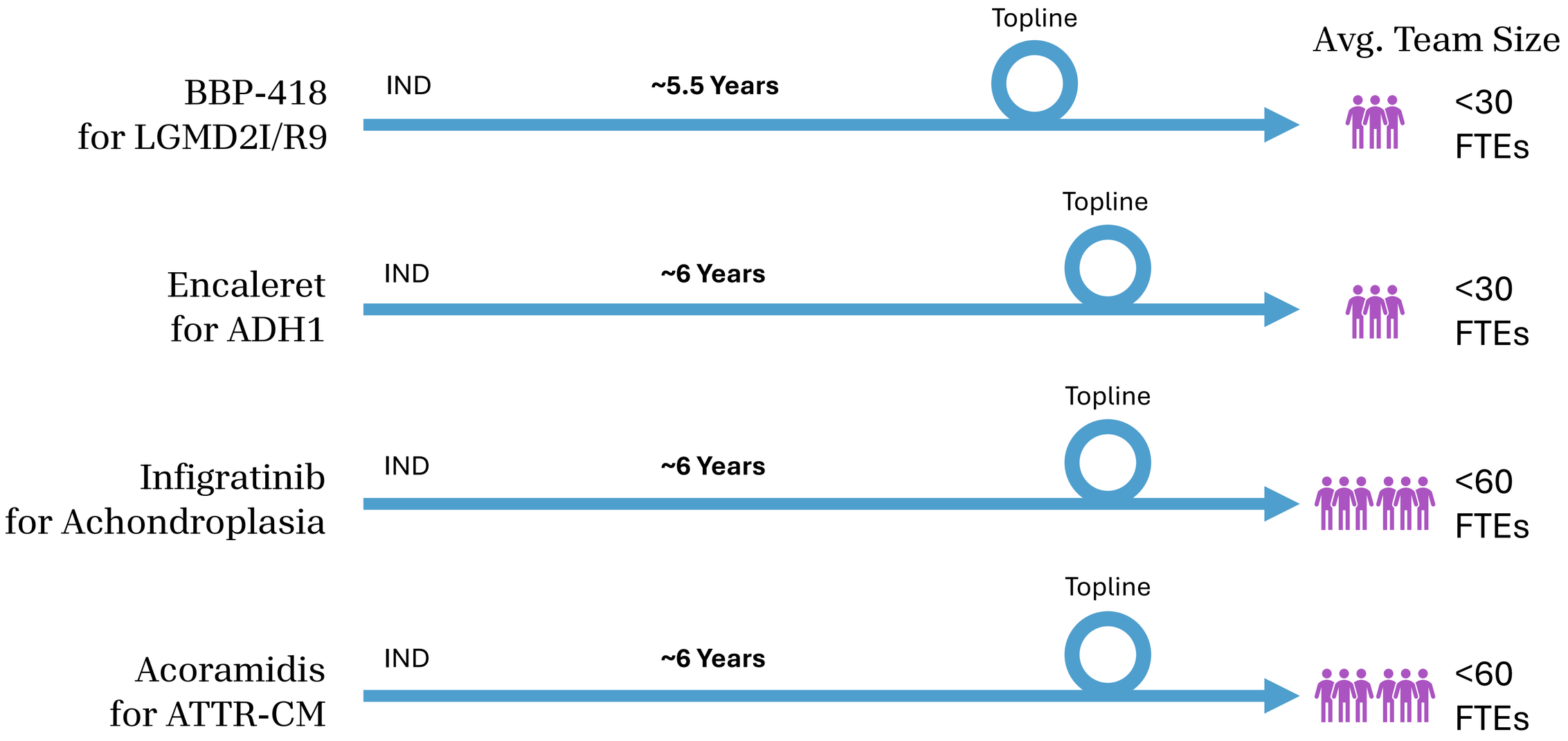
3 Positive Phase 3 Results

- fortify ✓
- CALIBRATE ✓
- PROPEL3 ✓
- CANaspire
- ACCEL 2/3
- RECLAIM-HP

15	> 70	> 35	19	< $40M
active trials in ecosystem	papers published	academic partnerships	INDs created	spend to proof-of-concept data

Fortify results reflect interim analysis data. BridgeBio does not commercialize Nulibry but receives royalties. Truseltiq was withdrawn from the market voluntarily after receiving FDA approval.

We leverage lean operating teams that advance medicines quickly and efficiently for people living with rare diseases



Source: internal estimates. IND: Investigational New Drug application; FTE: Full Time Employee.

A pipeline of products that sing across the BridgeBio ecosystem

Program	Indication	Pre-Clinical	Phase 1	Phase 2	Phase 3	Approved	Patients (US + EU)	Market Opportunity
Attruby (acoramidis)	Transthyretin Amyloidosis (ATTR-CM)					✓	500,000+	$20B+
Attruby (acoramidis)	Hereditary Transthyretin Amyloidosis (hATTR-PN and hATTR-CM) Prevention						~50,000	$1B+
Nulibry (fosdenopterin)	Molybdenum Cofactor Deficiency (MoCD) Type A					✓	<100	Partnered
Infigratinib	Achondroplasia (ACH)					Topline Readout	55,000+	$2B+
	Hypochondroplasia (HCH)				Observational Run-in Fully Enrolled for Ph. 2		55,000+	$2B+
BBP-418	Limb-Girdle Muscular Dystrophy Type 2I/R9 (LGMD2I/R9)					PDUFA Nov 27 2026	7,000+	$1B+
Encaleret	Autosomal Dominant Hypocalcemia Type 1 (ADH1)					NDA Submitted	25,000+	$1B+
	Chronic Hypoparathyroidism (HP)						200,000+	$1B+
BBP-812	Canavan Disease				Phase 1/2 Pivotal		1,000	TBD
Evanesco (depleter)	Transthyretin Amyloidosis (ATTR-CM)						500,000+	$20B+
GondolaBio	17 Programs (various indications)						Various	Various
EPP	Erythropoietic Protoporphyria						25,000	TBD
ADPKD	Autosomal Dominant Polycystic Kidney Disease						300,000	TBD
AATD	Alpha-1 Antitrypsin Deficiency						200,000	TBD
BridgeBio Oncology Therapeutics	3 Oncology Programs (various indications)						Various	Various

As of March 31, 2026, BridgeBio has an 18.2% ownership stake in BridgeBio Oncology Therapeutics and a 20.4% ownership stake in GondolaBio. BridgeBio Oncology Therapeutics and GondolaBio are independent companies from BridgeBio. BridgeBio's interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded.

BridgeBio's portfolio at a glance

	Key Data	Peak Sales	Peak Share
ATTR-CM	▪ Effect seen as early as 1 month, 42% reduction in all cause mortality and cardiovascular hospitalization at Month 30, 50% reduction in cumulative frequency of CV hospitalizations at Month 30 ▪ First and only near complete stabilizer of TTR ▪ Best in class point estimates in key sub-populations like variant and Afib	>$4.0B	~30%
ACH	▪ Treatment effect: 2.10 cm / year (p<0.0001); 5.96 cm/yr absolute AHV on treatment ▪ Proportionality: -0.05 vs Pbo in ages 3-8 (p<0.05; first stat sig effect in PBO-controlled trial) ▪ Clean safety (Only 3 cases (4%) of mild, asymptomatic and transient elevations of phos, no issues with hypotension or hypertrichosis); positioned to be the first oral option	>$2.0B PRV eligible	~65%
ADH1	▪ Serum / urine calcium normalization: 76% at Week 24 (p<0.0001) ▪ Intact PTH above lower limit of reference range: 91% at Week 24 (p<0.0001) ▪ Well-tolerated and oral	>$1.0B Priority review eligible	First and only
LGMD2I/R9	▪ Glycosylated αDG: 1.8x increase from baseline at 3 months (p<0.0001) ▪ All key secondary stat sig: Glycosylated αDG at 12 months (p<0.0001), Serum CK (p<0.0001), 100MTT (p<0.0001), FVC (p=0.0071) ▪ NSAD: stat sig improvement vs. PBO* ▪ Well-tolerated and oral	>$1.0B PRV eligible	First and only

Note: * Nominally statistically significant based on exploratory analysis; analysis not part of alpha-controlled hierarchy at interim analysis

We are well-financed to hit a drumbeat of potential milestones in 2026

Cash, Cash Equivalents and Marketable Securities

BridgeBio currently has $940.2M[1] in cash, cash equivalents and marketable securities

Convertible Notes Offering

In January 2026, BridgeBio raised an additional $619.3M in capital through a convertible senior notes offering

Cash Flow Expectations

BridgeBio expected to be significantly cash flow positive in 2028

1H 2026

- **Infigratinib:** ACH Topline
- **Encaleret:** Initiate P2/3 pediatric ADH1
- **BBP-418:** NDA submission
- **Encaleret:** NDA submission

2H 2026

- **Encaleret:** Initiate P3 CHP trial
- **Infigratinib:** NDA submission
- **Infigratinib:** HCH P2 data update

1H 2027

- **BBP-418:** FDA approval and product launch
- **Encaleret:** FDA approval and product launch

1. Cash, cash equivalents and marketable securities number as of March 31, 2026

Centralized scale + disease-level focus = ability to have multiple, focused launches

Benefits of centralized commercial model

Build once, scale many
Build commercial capabilities once and leverage across launches

Cost synergies
Leverage fixed costs; Limited incremental additional costs per rare disease launch

Compound launch expertise
Apply cross-launch insights in both competitive and first-in-class markets to systematically de-risk execution

Benefits of execution at the decentralized program level

Deep program expertise
Dedicated affiliate teams stay with programs over time, building deep clinical and community knowledge

Tailored launch strategies
Each program can pursue the right access, positioning, and sequencing approach

Sharper execution
Smaller, specialized markets benefit from tailored approaches

Attruby®
Approved for ATTR-CM

Key Attruby® (acoramidis) highlights

Attruby® (acoramidis) 356 mg tablet

First and only approved product with a label specifying **near-complete stabilization** of TTR

Effect seen as early as 1 month – reduction in cumulative cardiovascular outcomes within the first month of treatment in patients with ATTR-CM

42% reduction in composite of all-cause mortality and recurrent cardiovascular-related hospitalization events at Month 30

50% reduction in the cumulative frequency of cardiovascular-related hospitalization events at Month 30

43% reduction in frequency of CVH due to atrial fibrillation

$180.6M	>25%	30-40%	$20B
Q1 2026 Net Product Revenue	Estimated Share of NBRx for Attruby	Potential Peak Market Share for Attruby	Potential Peak ATTR Market Sales for All Products

Commercial statistics as of February 2026. Peak market share based on survey of 200 HCPs with a history of ATTR-CM prescribing in Rx data. Consensus estimates of $15-20B ATTR market sales at peak with $9B in 2025.

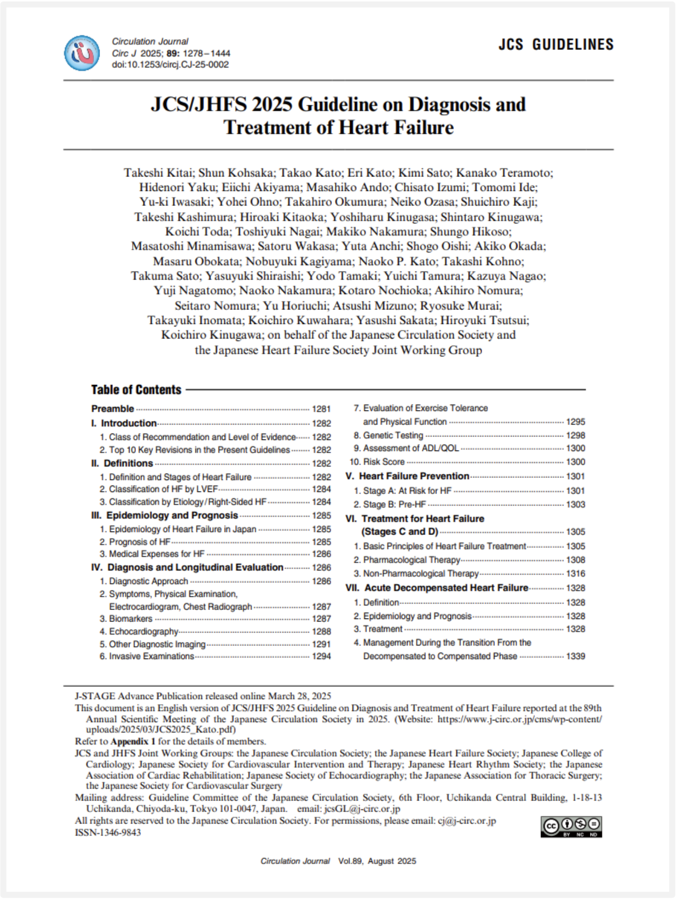
Beyonttra® (acoramidis) has seen rapid EU uptake with leading guideline endorsements

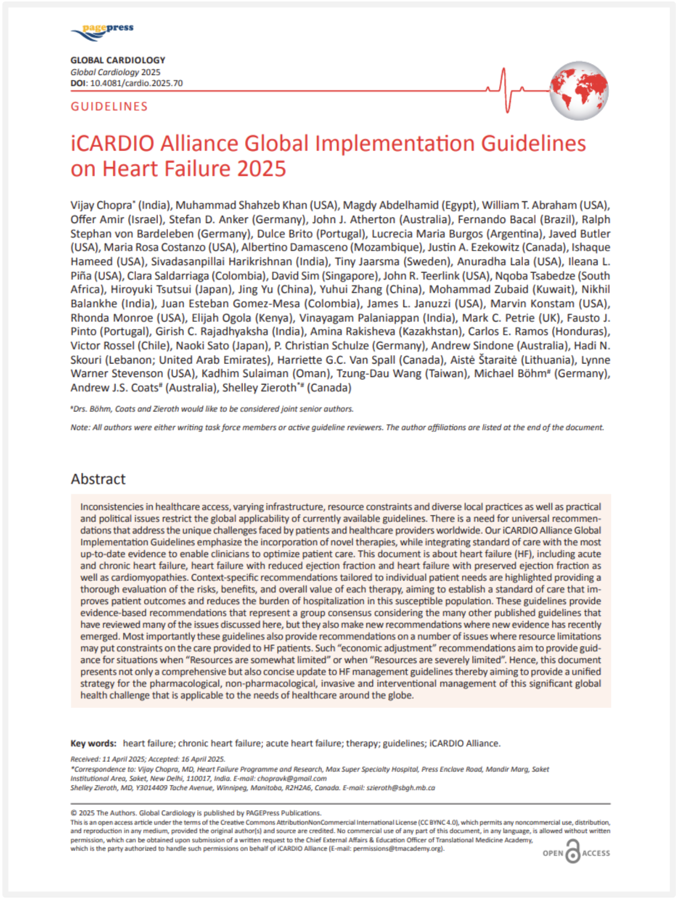
BEYONTTRA®
(acoramidis) 356mg tablet

50%
Germany NBRx share 8 months into launch

98%
Denmark volume share

Global Guidelines Endorse Beyonttra (acoramidis) for Clinical Confidence, Credibility, and Impact


JCS GUIDELINES
JCS/JHFS 2025 Guideline on Diagnosis and Treatment of Heart Failure


GUIDELINES
iCARDIO Alliance Global Implementation Guidelines on Heart Failure 2025


Revista Portuguesa de Cardiologia
GUIDELINES
Portuguese recommendations for the management of transthyretin amyloid cardiomyopathy (Part 1 of 2): Screening, diagnosis and treatment. Developed by the Task Force on the management of transthyretin amyloid cardiomyopathy of the Working Group on Myocardial and Pericardial Diseases of the Portuguese Society of Cardiology
Recomendações Portuguesas da Amiloidose Cardíaca ATTR (parte 1 de 2): Rastreio, diagnóstico e tratamento. Desenvolvidas pelo Grupo de Trabalho para as recomendações da ATTR-CM do Grupo de Estudos das Doenças do Miocárdio e do Pericárdio da Sociedade Portuguesa de Cardiologia

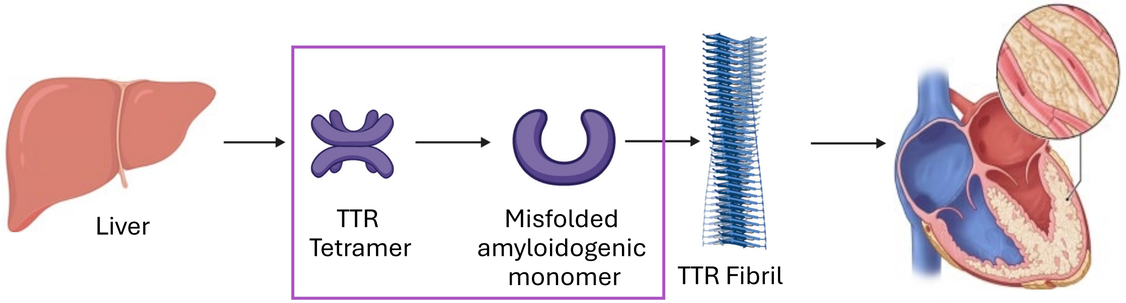
Beyonttra (acoramidis) demonstrates robust commercial uptake in Germany, suggesting strong uptake in other 15 EU countries

Acoramidis endorsed with highest tier recommendation and highlighted as near-complete stabilizer while tafamidis described as partial stabilizer

Beyonttra is partnered with Bayer in the EU. JCS/JHFS 2025 Guideline; J Card Fail. 2025 Mar 27; S1071-9164(25)00100-9; iCARDIO Alliance HF 2025, Chopra V. et al., Heart Lung Circ 34(7):e55–e82. Marques N, Aguiar Rosa S, Cordeiro F, et al.. Rev Port Cardiol. 2025;44(1):7-48. doi:10.1016/j.repc.2024.12.002. IQVIA™ LRx (Germany). NBRx share for acoramidis; period ending August 31 2025, reporting 15 Oct 2025.

Attruby's advantages stem from the fact that it is the only near-complete stabilizer, as more TTR is associated with a longer life

ATTR-CM is a progressive disease of TTR protein instability



Attruby: near-complete stabilizer that prevents dissociation of TTR tetramers into monomers

- **TTR protein instability:** normally stable TTR tetramers dissociate into instable monomers
- **Cardiac infiltration:** Instable TTR monomers misfold and deposit in as insoluble amyloid fibrils in the heart and nerves
- **Cardiovascular risk:** Increased risk of cardiovascular-related mortality (CVM) and cardiovascular-related hospitalizations (CVH)

Attruby uniquely achieves near-complete stabilization, preserving the important TTR protein

Attruby is **the only** TTR stabilizer with near-complete (≥90%) stabilization reflected in its **approved label**

How Attruby achieves near-complete stabilization

- **Sees more target** (superior free fraction)
- **Binds more target** (superior K_D2)
- **Glues the target together stronger** (enthalpic binding mode)

Why near-complete stabilization matters

- **More TTR, longer life:** Higher serum TTR independently predicts improved survival[1 2]
- **Every increment counts:** Each 5 mg/dL rise in serum TTR associated with ~32% lower odds of death by 30 months[2]

Nelson et al., Amyloid 2020; 28(1), 24–29. Miller, M. et al. J Med Chem. 2018;61:7862-7876. Pinheiro, F. et al. Proc Natl Acad Sci U.S.A. 2026; 123(1) e2519908122. AG10 is the former internal development code for Attruby/Beyonttra (acoramidis). 1Christoffersen, M. et al., JAMA Cardiology, 2024 2Maurer et al. Early Increase in Serum Transthyretin by Acoramidis Independently Predicts Improved Survival in TTR Amyloid Cardiomyopathy (JACC 2025)

Attruby delivers benefits in high-risk variant patients and reduces atrial fibrillation-related adverse events

Outstanding outcomes in ATTRv-CM patients

V122I ATTRv-CM has median survival of just ~31 months vs. ~60 months in ATTRwt-CM — a historically poor-prognosis population.[1]

Statistically significant benefit with 0.41 hazard ratio on composite ACM or first CVH in ATTRv-CM participants vs. placebo[2]

Population	N (%)	Hazard Ratio (95% CI)	p-value
Overall Population	611 (100%)	0.65 (0.50-0.83)	0.0008
ATTRv-CM	59 (9.7%)	0.41 (0.21-0.81)	0.0109
V122I	35 (5.7%)	0.31 (0.12-0.81)	0.016

Unprecedented gains in QOL and functional capacity in ATTRv-CM[3]

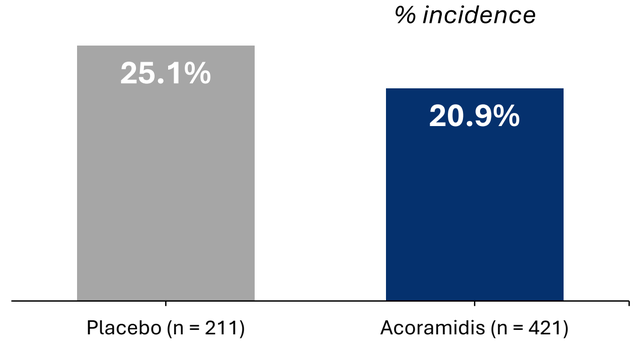
6MWD
+86.7 m
vs. placebo · p = 0.0048

KCCQ-OS
+20.3 pts
vs. placebo · p = 0.0019

Reduced atrial fibrillation burden[4]

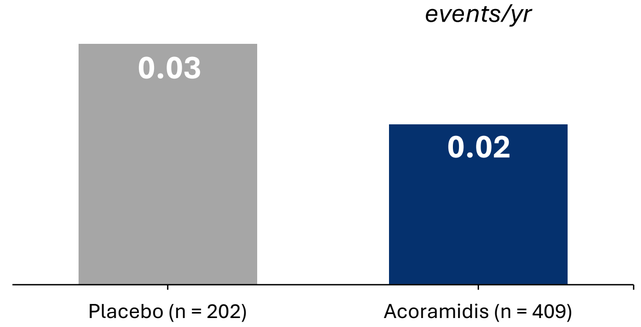
Incidence of AF adverse events

17%
relative risk reduction vs. placebo



Annual frequency of CVH due to AF

43%
relative risk reduction vs. placebo



Note: AF, atrial fibrillation; AFL, atrial flutter; AF-type adverse event covers the MedDRA preferred terms Atrial fibrillation, Atrial flutter, and Cardiac flutter.

Attruby Demonstrates Improved Real World Clinical Outcomes vs. Tafamidis

Real world evidence suggests that Attruby achieves a 43% reduction in diuretic intensification relative to tafamidis

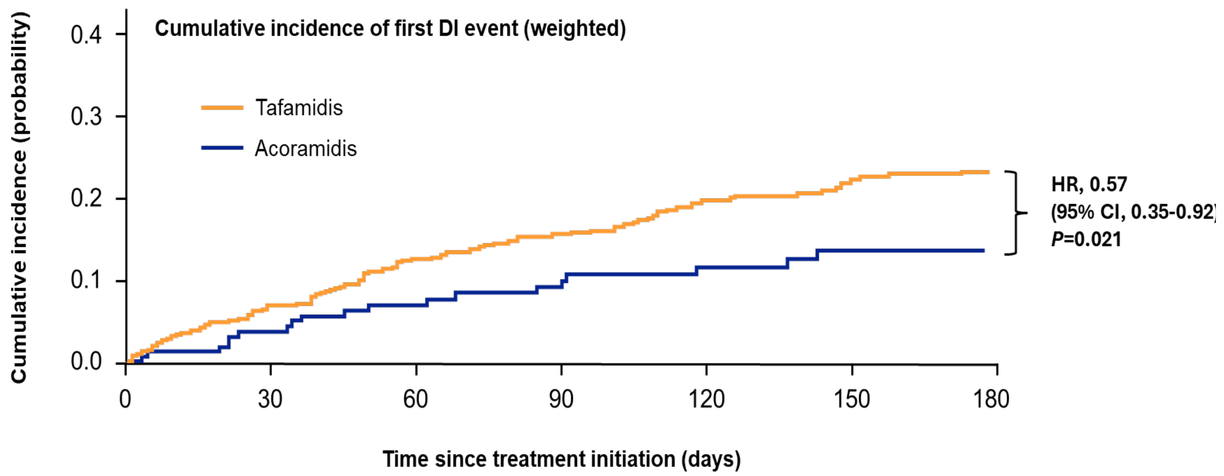
Acoramidis vs. Tafamidis · Propensity Score Weighted Analysis · N=170 vs. ESS=448 · Mean f/u: 140 days · SGLT2i use: ~44%

43%
Reduction in Diuretic Intensification

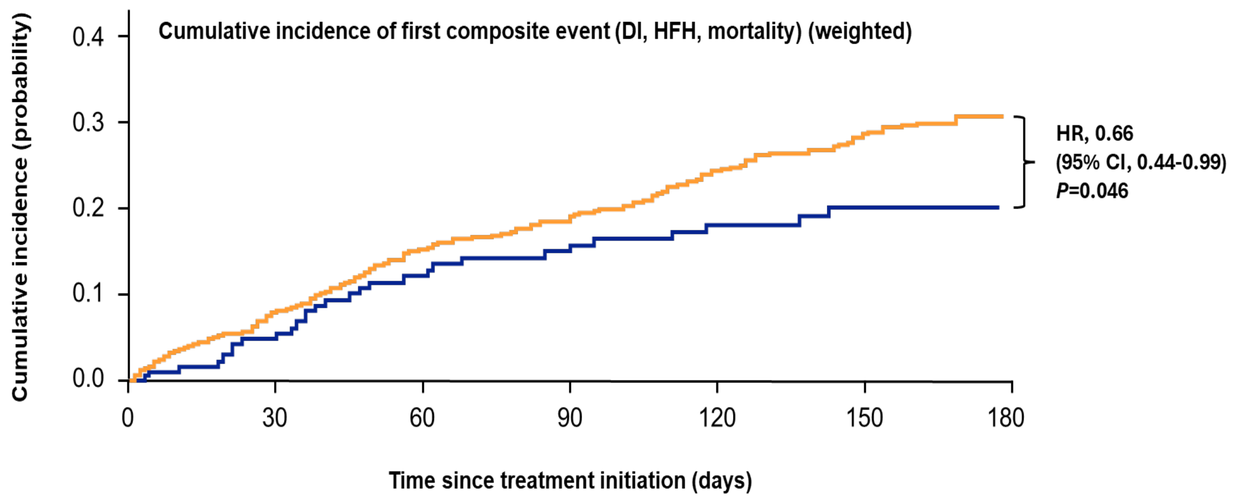
34%
Reduction in Composite Endpoint

Outcome	Acoramidis (N=170)	Tafamidis (ESS=448)	HR (95% CI)	p-value
A) Diuretic Intensification (DI)	20 (11.8%)	92 (20.5%)	**0.57 (0.35–0.92)**	**0.021**
B) DI + HF hospitalization + mortality	30 (17.6%)	118 (26.3%)	**0.66 (0.44–0.99)**	**0.046**

A



B

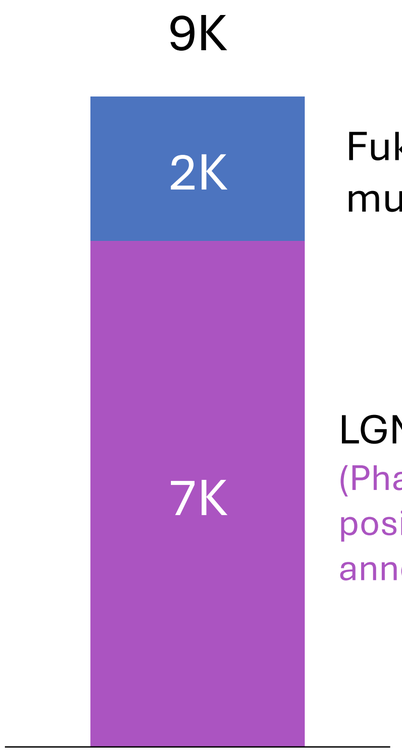


ESS = effective sample size; DI = diuretic intensification; Composite = DI + heart failure hospitalization + all-cause mortality;
Patient ID period: 12/11/2024–04/30/2025; study end: 07/31/2025. Wright, Martyn, Keshishian, et al. 2026.medRxiv.04.24.26351684.

BBP-418

LGMD2I/R9 is a progressive neuromuscular disease with high unmet need, representing a >$1B market opportunity in the US and EU

Addressable patients by indication



Fukuyama congenital muscular dystrophy *(Japan)*

LGMD2I/R9[1,2] *(US + EU)*
(Phase 3 fully enrolled and positive interim analysis topline announced as of Oct. 2025)

Unmet need

LGMD2I/R9 is an inherited neuromuscular disorder characterized by lower-limb weakness and loss of ambulation as well as respiratory decline and cardiac dysfunction

No approved therapies for LGMD2I/R9

Current **standard of care is aimed at symptom management** and includes physical therapy, cardiac and pulmonary function surveillance, and pain management

Standard of care does not prevent continuous and progressive decline in LGMD2I/R9 patients

Market opportunity $1B+ *program PRV eligible*

[1] Liu, et al. Genetics in Medicine. 2019. [2] Includes all patients with potentially treatable mutations in FKRP, FKTN, and ISPD.

Unprecedented and consistent improvement across primary and all key secondary efficacy endpoints combined with well-tolerated safety profile

Met primary and all key secondary endpoints vs. placebo in Phase 3 FORTIFY interim analysis

1.8x
increase in glycosylated αDG at 3 months

82%
decrease in CK at 12 months

Faster ambulation
compared to deterioration on placebo

Improved pulmonary function
compared to deterioration on placebo

Improved gross motor function
compared to deterioration on placebo*

Well-tolerated safety profile
including no discontinuations due to TEAEs

Consistent benefit across subgroups

1. L276I homozygous vs. Other *FKRP* genotypes
2. Pediatric (12-17 years) vs. Adult (18-60 years)
3. FVC at baseline (≥80% vs. 40-80%)

Favors BBP-418 vs. placebo across all subgroups for all key efficacy endpoints

Potential for therapeutic cure

Some patients achieved normal levels on key biomarkers in 1 year
including 38% of patients with CK within upper limit of normal

Note: * Nominally statistically significant based on exploratory analysis; analysis not part of alpha-controlled hierarchy at interim analysis; αDG = alpha dystroglycan; CK = creatine kinase; FVC = Forced Vital Capacity
Source: Data on file

We completed a successful meeting with FDA post interim analysis, and they recommend orienting our NDA toward traditional approval

Oct. 2025

- Positive topline results from Phase 3 FORTIFY study interim analysis

Dec. 2025

- FDA acknowledged the data ***"...demonstrate consistent treatment effects on multiple efficacy endpoints..."***
- **FDA recommends orienting NDA toward traditional approval**

Mar. 2026

- Submit New Drug Application (NDA) to FDA

2026

- Engage regulatory agencies in Europe

U.S. Approval and Commercial Launch in LGMD2I/R9

Anticipated Late 2026 / Early 2027

Leveraging our proven commercial infrastructure to successfully launch BBP-418

Built on the foundation of Attruby's commercial success and tailored to the unique LGMD2I/R9 market opportunity

Established commercial infrastructure

Dedicated commercial and medical leadership onboarded; field team sized consistent with comparable rare neuromuscular launches, focused on the concentrated prescriber base at

Proven launch playbook

NDA submitted to FDA; Fast Track Designation supports potential Priority Review; U.S. launch anticipated late 2026/early 2027

Ensure broad access

Leverage first-in-class data to secure broad coverage; if approved, BBP-418 would be the first approved therapy for any form of LGMD

We will commercialize and launch BBP-418

Our commercial strategy combines proven rare disease launch capabilities with engagement tailored to neuromuscular specialists and the LGMD2I/R9 community with a field team sized to match the concentrated prescriber base at MDA centers

Strategic area	Objective
Position BBP-418 as the standard of care	FORTIFY met all primary and secondary endpoints at interim; full dataset presented at 2026 MDA Conference
Systematic patient identification	~500 genetically confirmed LGMD2I/R9 patients identified through account profiling, genetic testing databases, registry data, and clinical site mapping
Drive awareness and accelerate diagnosis	Focused on concentrated prescriber base at MDA care centers; actively uncovering undiagnosed patients within broader LGMD and Becker MD populations
Build urgency for early diagnosis and initiation	KOL engagement ongoing: European centers see higher patient concentration; U.S. care more distributed, reinforcing targeted field deployment model

Encaleret

There are no therapies currently indicated to treat ADH1, a serious and rare genetic condition in a >$1B market

US Market Opportunity

12K Individuals with ADH1
~12K ADH1 prevalent population in the US based on 4 population databases[1-2]

9K Symptomatic
73% of patients with ADH1 are symptomatic[3]

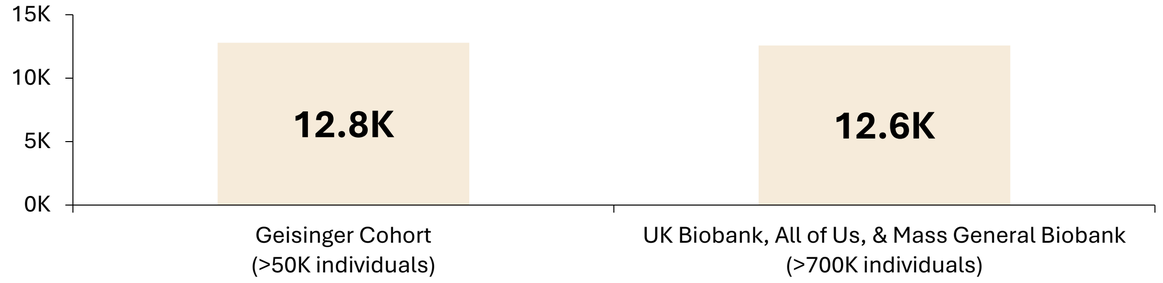
3K – 5K Currently Addressable
40-60% of symptomatic ADH1 patients are estimated to be diagnosed today; anticipate increase

~Nearly 2K US Patient Diagnosed with ADH over a 30-Month Period[6]

An analogous ADH1 market is XLH

	XLH	ADH1
Prevalence (US)	12K[4]	12K
Disease burden	Hypophosphatemia	Acute - hypocalcemia Chronic - hypercalciuria
Standard of care	Vitamin D, daily phosphate	Vitamin D, daily calcium
Registrational endpoint	Serum phosphate	Serum and urine calcium
Projected peak year sales	$2B+[5]	$1B+

ADH1 variant frequency estimates in literature[1-2]



Encaleret directly targets ADH1 at its source

[1]Dershem, et al. Am J Hum Genet., 2020. [2]Chang, et al. Am J Hum Genet., 2025. [3]Roszko, et al. JBMR. 2022. [4]Dahir, et al. Jour Endo Soc., 2020. [5]Evaluate Pharma. [6]Komodo Claims Data, October 2023 – September 2025.
XLH = X-linked Hypophosphatemia.

CALIBRATE achieved & exceeded all criteria set forth as an upside target, with a 76% responder rate following 24 weeks of encaleret treatment

Upside Target Clinical Profile	Statistically significant primary analysis result compared to conventional therapy	At Week 24, ≥50% of study participants achieve target serum and urine Ca on encaleret	Majority of participants randomized to encaleret able to remain independent from conventional therapy[1]	At Week 24, mean iPTH within normal range on encaleret	Comparable safety and tolerability profile to conventional therapy
Outcome Observed	Primary endpoint met ($p < 0.0001$) demonstrating superiority to conventional therapy	76% (34 out of 45) achieved target serum and urine Ca on encaleret vs. 4% on conventional therapy	Among encaleret responders at Week 24, none required conventional therapy during Period 3[1]	Among encaleret responders at Week 24, none required conventional therapy during Period 3[1]	Encaleret was well-tolerated; no discontinuations related to study drug

[1]Requirement for conventional therapy defined as oral calcium >600 mg/day and/or active vitamin D during Period 3.
Ca = Calcium; iPTH = Intact Parathyroid Hormone. Encaleret is an investigational drug. Its safety and efficacy have not been fully evaluated by any regulatory authority.

Encaleret also has potential as an oral medication for other etiologies of chronic hypoparathyroidism (HP) representing a >$1B market opportunity

Chronic Hypoparathyroidism (~200K in US+EU)[1,2,3]

Encaleret expansion opportunity (RECLAIM-HP trial)

- **Post-surgical (65%-75%)[4,5]**
- **Genetic (~12.5%)[5]**
  - **ADH1**
- **Autoimmune (<10%)[5]**
- **Idiopathic (<10%)[5]**

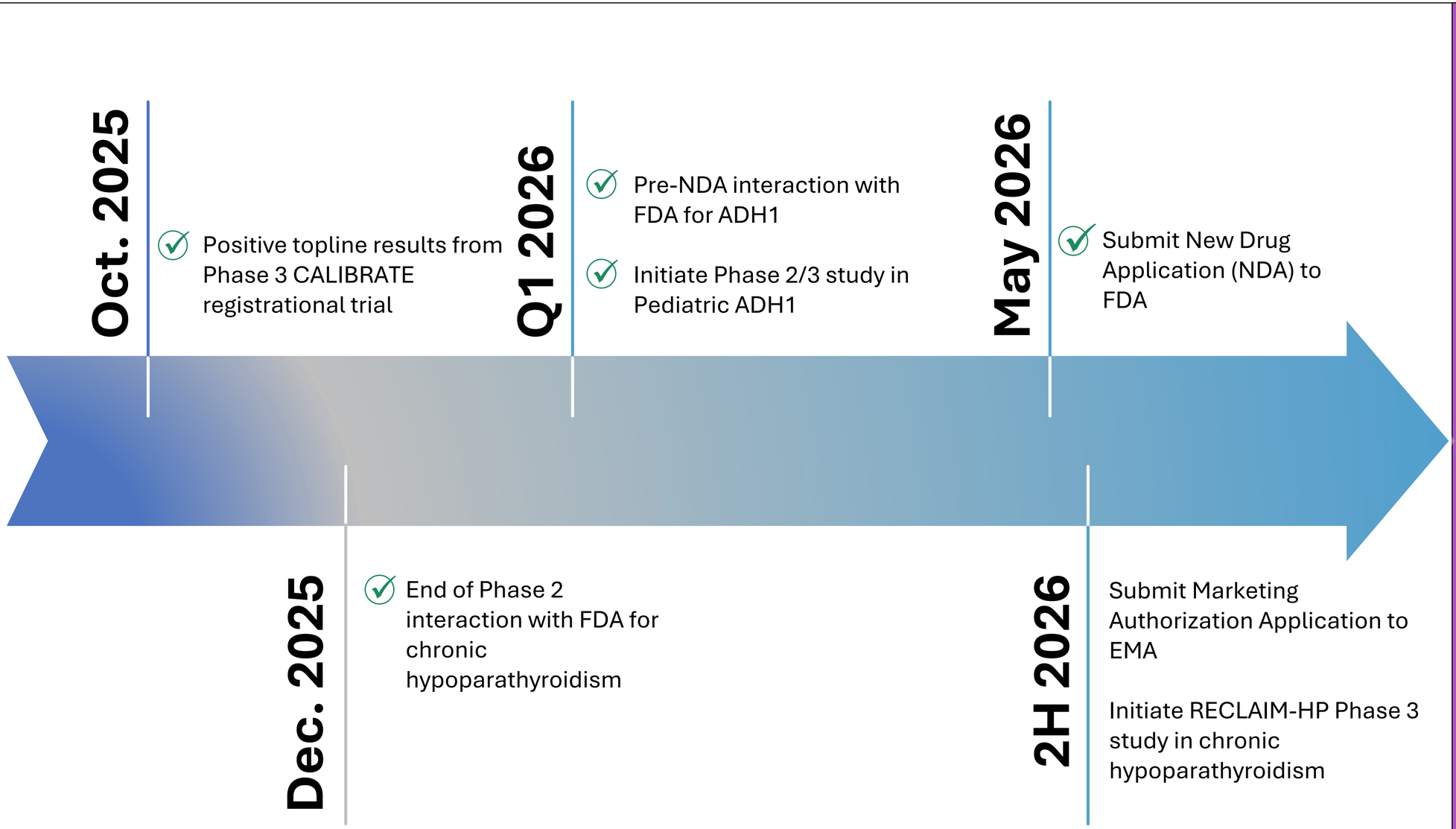
Encaleret current development focus and registrational opportunity

CALIBRATE PEDS

CALIBRATE

[1]Powers et al., J. Bone Miner. Res., 2013. [2]Clarke et al., J. Clin. Endocrinol. Metab., 2016. [3]Underbjerg et al., J. Bone Miner. Res., 2013. [4]Mannstadt et al., Nat. Rev. Dis. Primers., 2017. [5]Kiam et al., Clin. Endocr., 2022.

NDA submission for ADH1 planned in the first half of 2026 with two additional registrational studies to initiate this year



U.S. Approval and Commercial Launch in ADH1
Anticipated Early 2027

Leveraging our proven commercial infrastructure to successfully launch encaleret

Built on the foundation of Attruby's® global commercial success and tailored to the unique ADH1 market opportunity

Established commercial infrastructure
Dedicated commercial & medical leadership onboarded
Encaleret field teams supported by home office leadership built around the Attruby launch

Proven launch playbook
Applying Attruby launch learnings to an ultra-rare, first-in-class setting;
NDA submission on track for 1H 2026 with U.S. launch anticipated early 2027

Targeted resource deployment
Team built with focus on rare genetic disease and endocrinology/nephrology launch experience
KOL partnerships and patient ID programs already active

How we will win

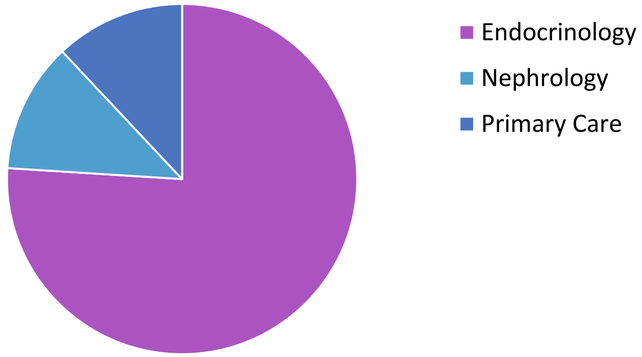
Redefining standard of care in ADH1

Strategic area	Objective
Capitalize on potential first-in-class profile	**Position encaleret as the new standard of care in ADH1** CALIBRATE primary results to be presented at ECE (Prague, May 2026)
Disease awareness	**Drive recognition of ADH1 as a genetically distinct condition** ~2,000 patients assigned ADH1 ICD-10 code (E20.810) since Oct 2023
Speed time to treatment	**Build urgency for early diagnosis and therapy initiation** Recently sponsored 3 U.S. family genetic testing events for known ADH1 probands; given autosomal dominant inheritance, ~50% of kindreds estimated to be affected
Maximize adherence	**Support patients and families through titration and long-term maintenance** CALIBRATE-PEDS enrolling first interventional study in children with any form of hypoparathyroidism; first cohort (ages 12–18) filled within weeks; some pediatric participants are children of CALIBRATE adults
Enable broad and global access	**Secure payer support and remove barriers to care globally** NDA on track for 1H 2026; MAA to EMA planned for 2H 2026

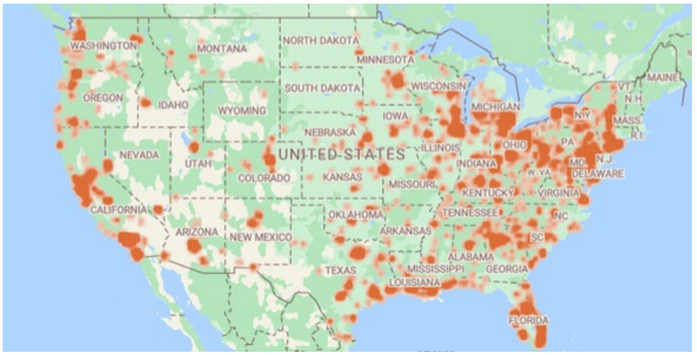
Data-driven commercial precision to accelerate ADH1 diagnosis and treatment

State of Market

ADH1 patients predominantly managed by endocrinologists



Concentration of HCPs Managing Patients



Concentrated prescriber base enabling targeted engagement

How we will capitalize on this first-in-category opportunity

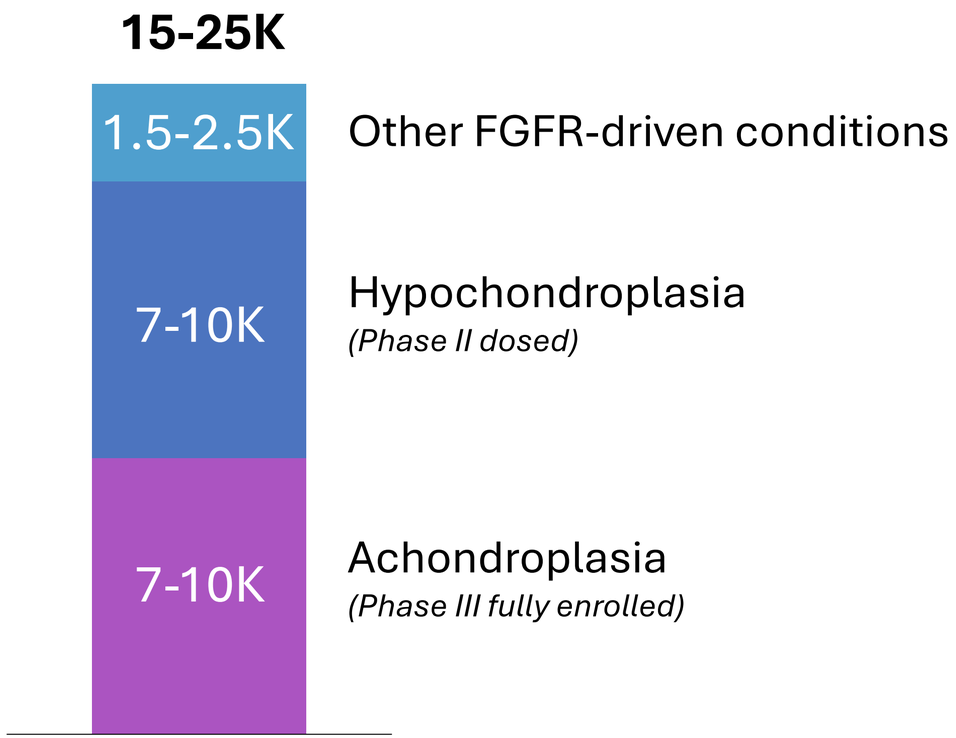
- **Leverage existing data to prioritize and activate top endocrinologists and nephrologists**
- **Employ AI/ML analytics to identify undiagnosed patients and managing HCPs**
- **Attract patients to diagnosis opportunities via familial screening and digital targeting**

Source: BBIO data on file; Symphony Health claims; Definitive Healthcare.

Infigratinib

There remains a significant unmet need for many children with skeletal dysplasias; this represents a large and rapidly growing market of >$5B

Addressable people by indication in US/EU[1]
(current population eligible for treatment)



55,000
individuals with achondroplasia globally

Represents **diagnosed** and **addressable** ACH population with open growth plates

$5B+
potential global market

[1]CDC birth estimates; EU Eurostats birth estimates; Foreman, et al. Am J Med Genet. 2020.; Bober, et al. Gene Reviews. 2020.; Wenger, et al. Gene Reviews. 2020.; Al-Namman, et al. J Oral Biol Craniofac Res. 2019. [2]Achondroplasia market includes all approved drugs.

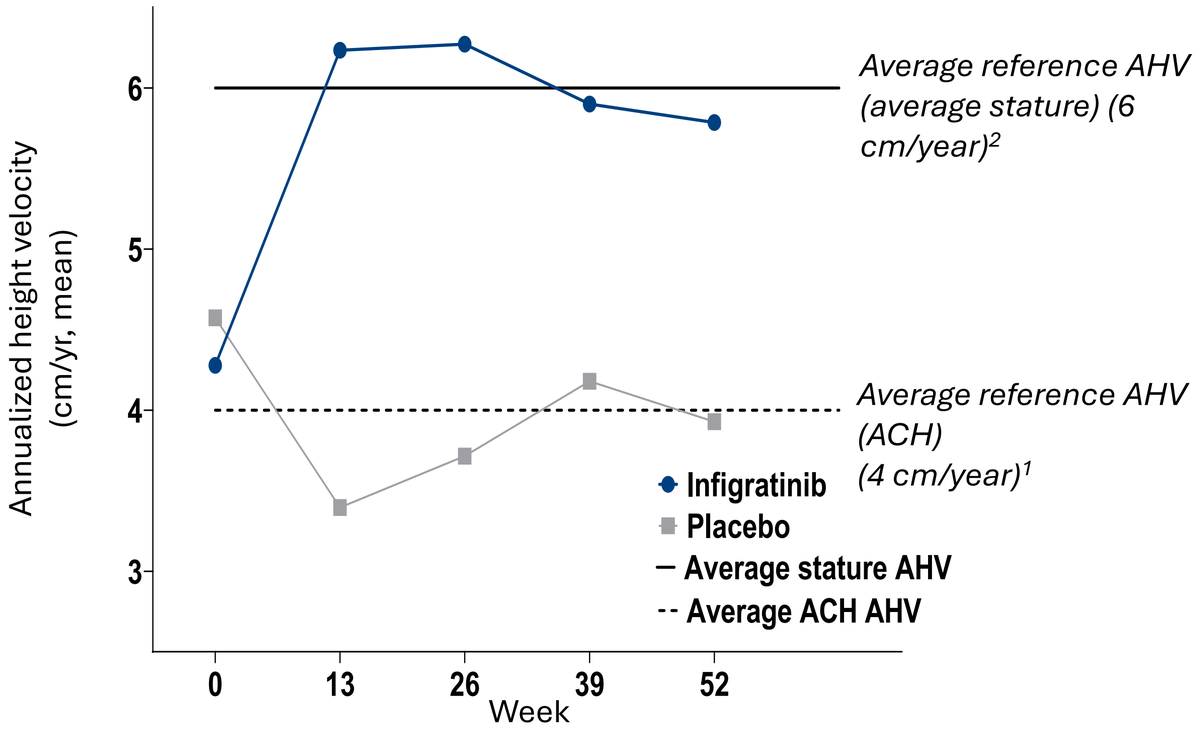
Infigratinib exceeded the bar for a potential best-in-class therapeutic option, in the broadest age range studied across any achondroplasia trial

Target Clinical Profile for Commercial Success	Outcomes
CFBL in AHV: More than +1.5 cm/yr against placebo at Week 52	**Met the primary endpoint** with mean difference against placebo of **+2.10 cm/yr** (p<0.0001), and a LS mean difference against placebo of **+1.74 cm/yr** (p<0.0001), the largest change observed in a randomized trial for ACH.
CFBL in height Z-score (ACH charts): More than +0.3 SD on treatment arm at Week 52	The LS mean improvement on the tx arm was +0.41 SD, the largest improvement observed in a RCT in ACH. LS mean difference against placebo was +0.32 SD (p<0.0001), the largest difference observed in a RCT in ACH.
Proportionality: More than 0.05 decrease in upper to lower body ratio on treatment arm	In a pre-specified exploratory analysis of children <8yrs of age (>50% of trial), there was a statistically significant LS mean change from baseline against placebo of -0.05 (p<0.05). In the overall population, infigratinib achieved a LS mean decrease of -0.05 on the tx arm, with a favorable LS treatment difference of -0.02 versus placebo at Week 52 (p=0.1849)
Safety: No symptomatic hypotension. Less than 10% low-grade hyperphosphatemia rate.	Well-tolerated safety profile, **consistent with no inhibition of FGFR1 and FGFR2**. ▪ No discontinuations related to study drug ▪ No serious adverse events related to study drug ▪ 3 cases of hyperphosphatemia (4%), all mild, asymptomatic, transient and did not require dose reduction or discontinuation ▪ No FGFR1 or FGFR2 associated AEs (E.g., retinal or corneal) ▪ No AEs associated with CNP analogues: symptomatic hypotension, ISRs, or hypertrichosis

BBIO data on file; Tx : treatment; pbo : placebo; ISR : injection site reactions; AEs : adverse events; RCT : randomized controlled trial.
**Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

Infigratinib improved growth to average stature levels with an oral therapy

Absolute Annualized Height Velocity (AHV)



Administration

Capsules (17 mm long)

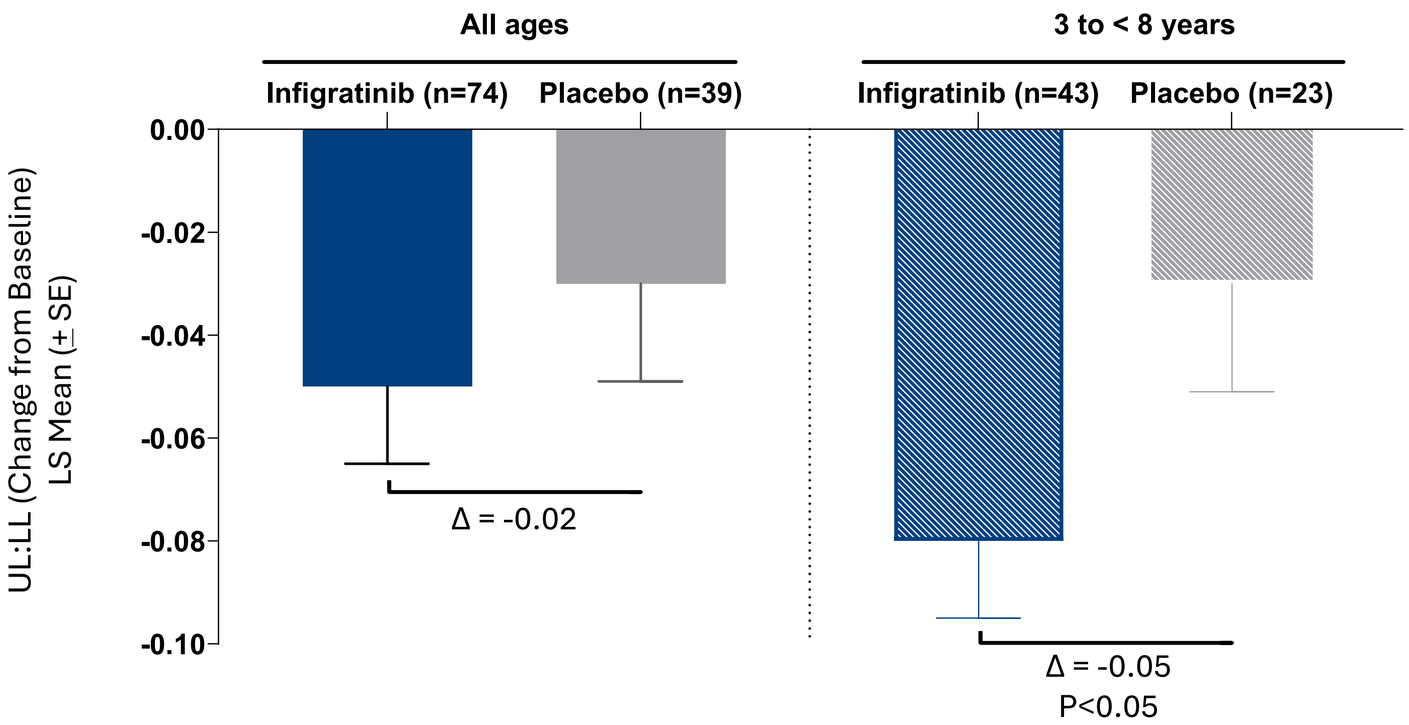
Minitablets (2 mm long)

Infigratinib demonstrated the largest absolute AHV (LS mean) at Week 52 in any randomized trial
Oral administration – no injections needed
Program has breakthrough designation and may be eligible for PRV upon approval

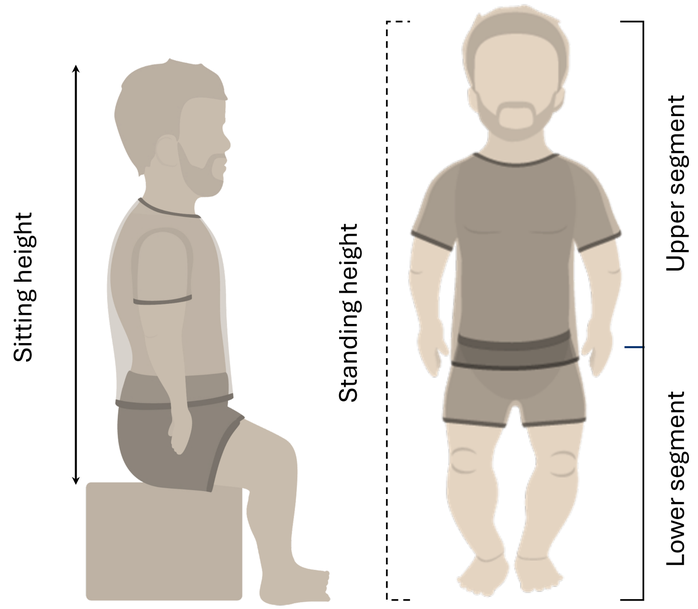
BBIO data on file; ACH: achondroplasia; 1-Hoover-Fong et al., 2021; 2-Sperling, 2020
**Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

Key secondary endpoint: Change from baseline to Week 52 in upper-to-lower body segment ratio

Change from baseline in upper to lower body segment ratio (UL:LL) at Week 52



Upper to lower body segment ratio is a measurable marker of disproportion in ACH



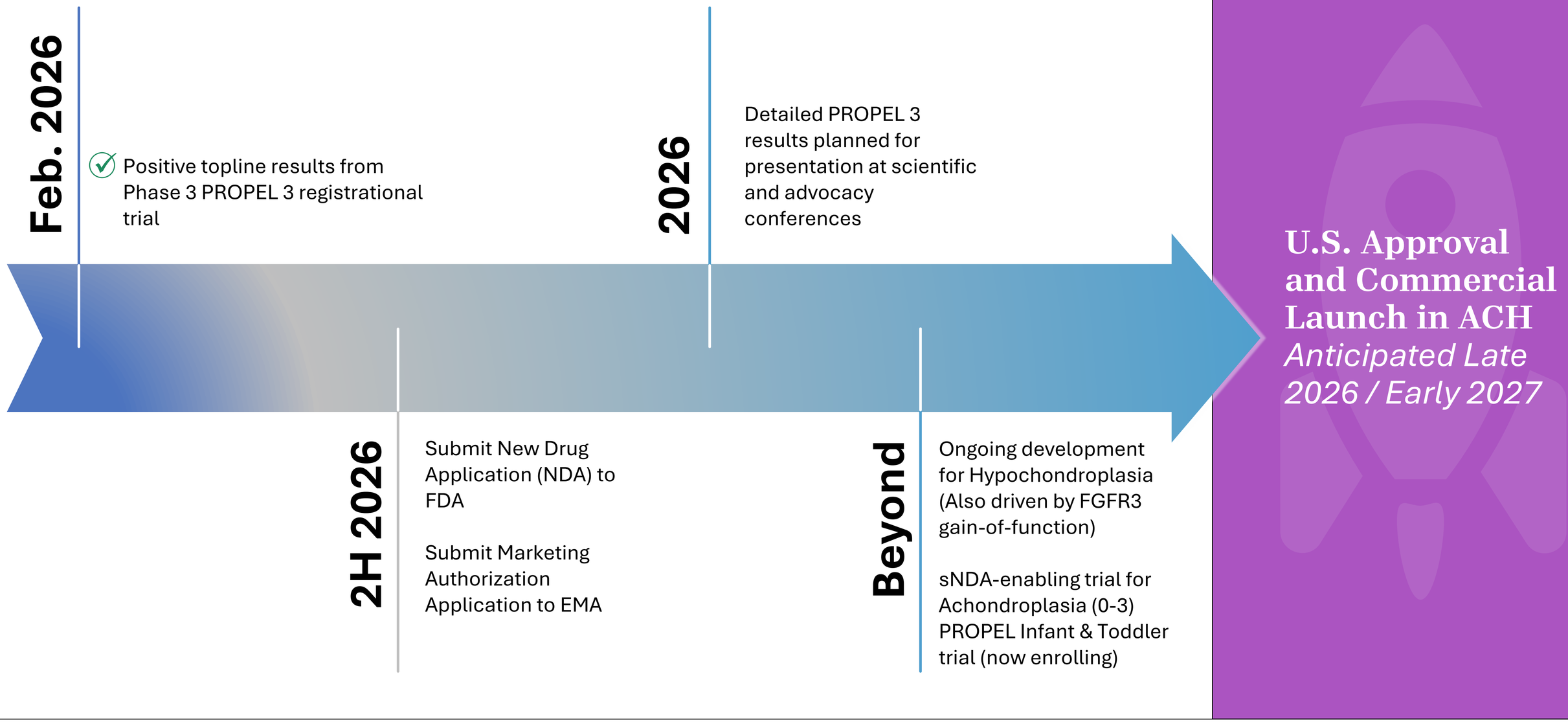
$$= \frac{\text{Sitting height}}{\text{Standing height} - \text{Sitting height}}$$

Infigratinib is the first therapeutic option to demonstrate a statistically significantly result on proportionality against placebo in a RCT for ACH

BBIO data on file; Exploratory analysis pre-specified in the SAP; RCT : randomized controlled trial

**Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

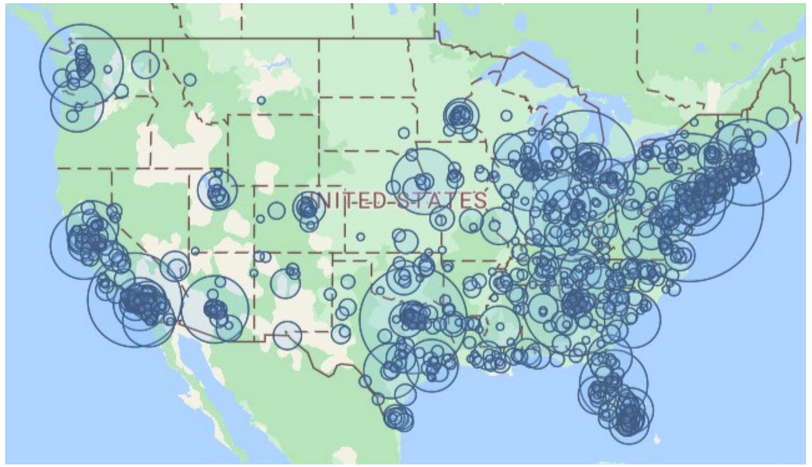
Infigratinib's NDA and MAA submissions in ACH expected in 2H 2026



Large concentrated market with global $5B opportunity

State of the market

Addressable market in the U.S.

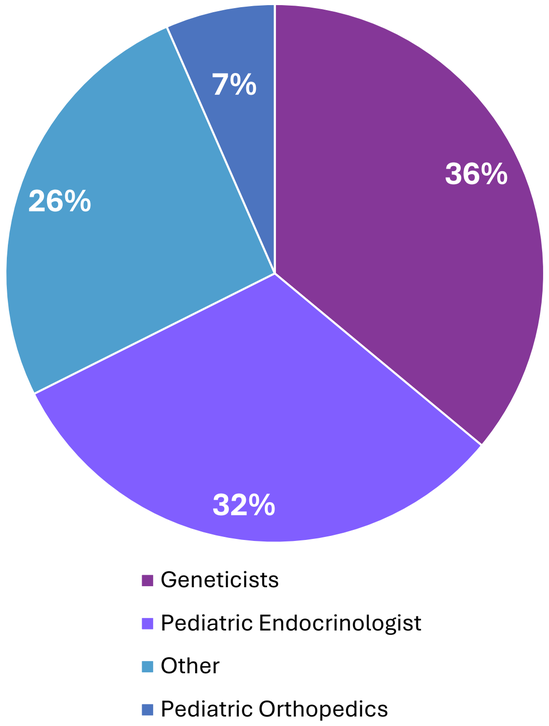


~2.9k

addressable individuals at launch in the U.S.

U.S. Prescriber base

People living with achondroplasia predominantly managed by geneticists & pediatric endos



We are preparing for a global launch

Infrastructure and capabilities in place to support a global launch

~55k

addressable individuals globally

Combining compelling data with a proven commercial platform

Key highlights

Best-in-class efficacy with largest AHV, height z score and **only stat sig proportionality improvement** to date across any ACH trial

Well-tolerated option, with no SAEs related to study drug or discontinuations due to AEs related to study drug

First- and only oral therapeutic option providing freedom from burden of daily or weekly injections, symptomatic hypotension or ISRs

World-class commercial team with proven launch and rare disease expertise

Strategic platform

	Strategic area	Objective
Commercial objectives	**Reinforce best in class treatment**	Establish **infigratinib** as the **first and only precision oral standard of care**
	Anchor FGFR3 as the core driver	FGFR3 inhibition is the validated biological foundation of ACH
	Accelerate treatment switching	Rapid conversion from injectables to a more convenient oral alternative
	Enable broad and global access	Secure payer support and remove barriers to care globally

BBP-812

Canavan disease is a severe, fatal, and ultra-rare neurodegenerative pediatric disease with no approved therapies

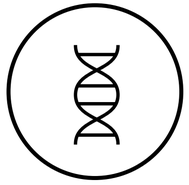
Unmet Need

~1,000 US and EU prevalence

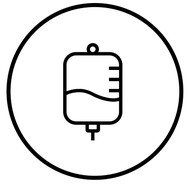
- Canavan disease (CD) is an ultra-rare neurodegenerative disease, usually fatal within the first two decades of life, and >25% of patients die by the age of 10 years
- Children with CD exhibit global and severe cognitive, motor, and language impairment, missing or regressing on most developmental milestones
- Children with CD require around the clock care – they cannot hold their heads up, sit, crawl, walk, are generally unable to speak, and suffer from seizures and spasticity
- There are no therapies available for Canavan disease

Design Principles

Provide first disease-modifying therapy
Target the condition directly at the source by directly replacing the mutated ASPA gene; utilize single registrational study & biomarker for accelerated approval



Provide therapy based on known safety profile
Leverage safety profile from approved IV AAV9 gene therapy



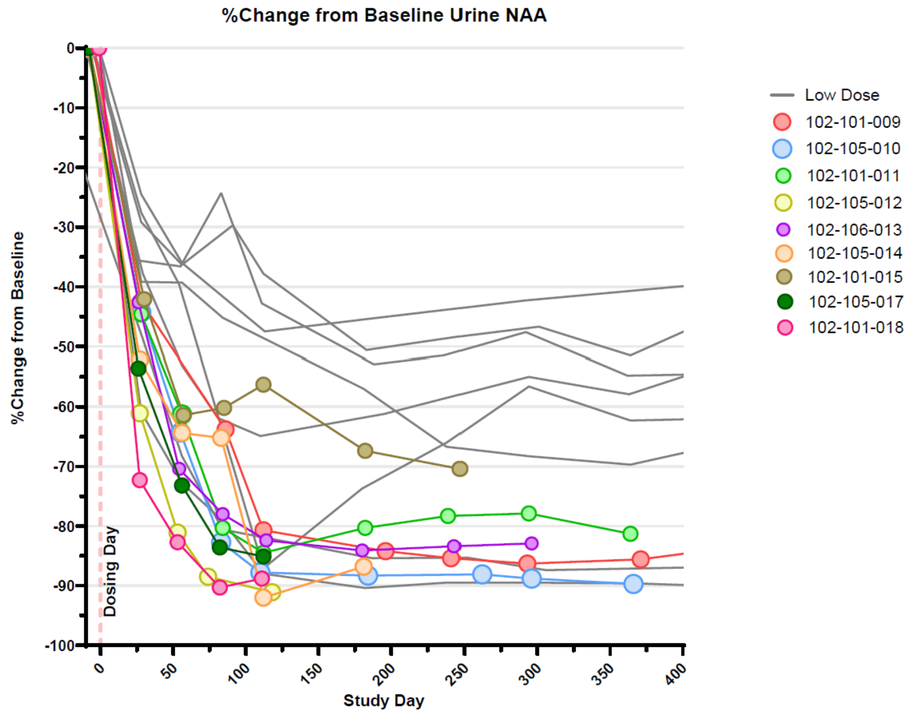
Avoid invasive neurosurgery
Provide a less invasive IV treatment option to minimize burden for patients and their caregivers

Program eligible for PRV upon approval

[1]Bley A, et al. Orphanet J Rare Dis. 2021 PMID: 34011350

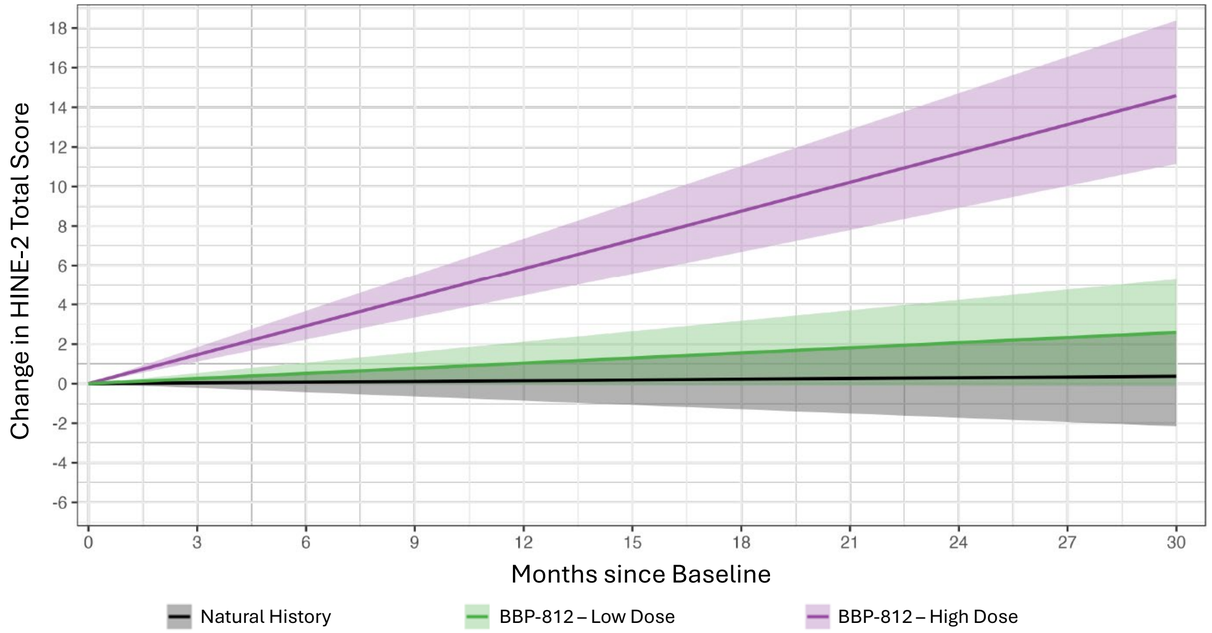
Current path to a potential BLA filing in 2027 based on reductions in urine NAA (surrogate endpoint) supported by motor function improvements

Urine N-acetylaspartic acid (NAA) levels



- **BBP-812 dose-dependently reduces urine NAA** to levels associated with only mild disease
- **FDA is open to the use of urine NAA as a surrogate endpoint** to support accelerated approval of BBP-812

Hammersmith Infant Neurological Examination (HINE-2) Trajectory



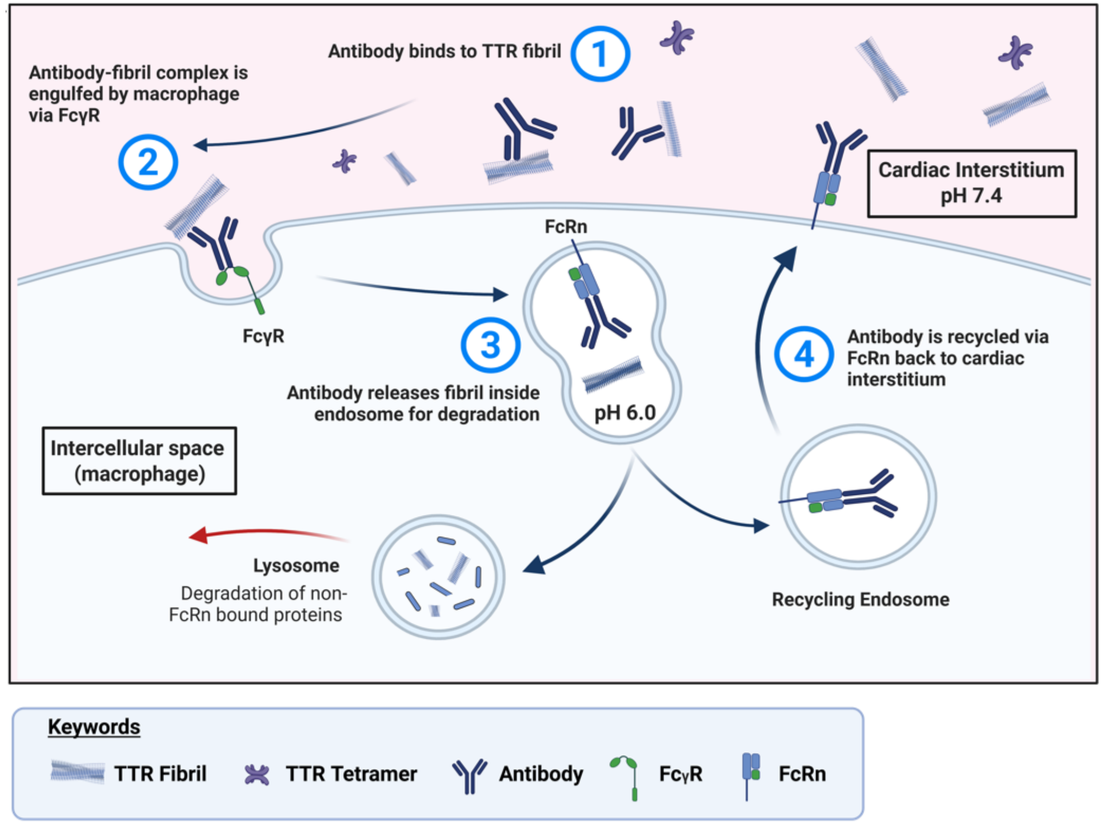
- Trajectory analysis shows **clear, dose-dependent separation in HINE-2 total score** with BBP-812 vs. natural history study
- Children are also showing **improvement on key motor metrics such as sitting, head control, and reaching / grasping**

Source: 10 December 2025 data cut, N = 18

Depleter Program

Guided by MOA, BridgeBio is engineering a differentiated depleter to explore the potential of ATTR-CM disease reversal

Depleter Mechanism of Action



BridgeBio's Differentiated Target Properties

1. **Improved fibril:tetramer binding ratio**
   - >10× preferential binding to misfolded TTR fibrils vs. native TTR tetramers

   ➔ **Binds more target**

2. **Faster macrophage recruitment**
   - First depleter to activate Fcγ receptors to boost macrophage activity

   ➔ **Clears more target**

3. **pH sensitivity**
   - Intentionally designed for pH-dependent antigen release inside macrophages

   ➔ **Extends antibody half-life**

4. **Half-life extension**
   - First depleter engineered for enhanced FcRn binding

   ➔ **Extends antibody half-life**

Depleter program expected to advance into the clinic in 2027–2028

MOA = Mechanism of action.

About Attruby® and BridgeBio

About Attruby® (acoramidis)

Attruby is the only near-complete (≥90%) stabilizer of Transthyretin (TTR) approved in the U.S. for the treatment of adult patients with ATTR-CM to reduce cardiovascular death and cardiovascular-related hospitalization. Attruby was generally well-tolerated. The most common side effects were mild and included diarrhea and abdominal pain that were resolved without drug discontinuation. BridgeBio offers an extensive suite of programs to help patients access our medicines. Visit Attruby.com for more information, including full Prescribing Information.

About BridgeBio Pharma, Inc.

BridgeBio Pharma, Inc. (BridgeBio) is a new type of biopharmaceutical company founded to discover, create, test, and deliver transformative medicines to treat patients who suffer from genetic diseases. BridgeBio's pipeline of development programs ranges from early science to advanced clinical trials. BridgeBio was founded in 2015 and its team of experienced drug discoverers, developers and innovators are committed to applying advances in genetic medicine to help patients as quickly as possible. For more information visit bridgebio.com.